

'The Bug Stops Here!' Nipah Encephalitis; Kerala 2019

Sir,
Nipah infection [NiV] recently claimed 17 lives in the Kerala Outbreak of May 2018.^[1] NiV is a negative-stranded RNA paramyxovirus and has a mortality rate of 40-75%. A 23-year-old man presented to our hospital with high grade fever, chills, and rigors for a duration of 10 days. On examination, he had fever, conjunctival congestion, drowsiness, dysarthria, severe ataxia, spontaneous and action induced myoclonus, tachycardia and tachypnea. Diffusion-weighted MRI [DWMRI] showed multiple tiny hyperintensities throughout the cerebral hemispheres, cerebellum and ponto-mesencephalic area with restricted diffusion ['Starry sky appearance'] [Figure 1].

Extensive serology and blood cultures were negative. Echocardiogram, Chest X-ray and ABG were normal. LP-CSF

on day 1 after admission showed 53 cells [Polymorphs 5%, Lymphocytes 95%] with normal sugar and protein. CSF [Acute encephalitic syndrome; AES panel [XCyton Diagnostics Ltd, Bengaluru] was positive for NiV on day 2. He was immediately shifted to a negative pressure isolation room and universal, standard droplet and bio-contaminant precautions were implemented. Real-time [RT-PCR], nested RT-PCR and anti-Nipah human IgM and IgG antibodies by ELISA were tested. He was started on oral Ribavirin for 11 days [Figure 2]. Contact identification and preventive measures were immediately initiated by the local health authorities. More than 300 people were placed under observation and 8 symptomatic contacts were quarantined. All of them tested negative. By day 21, all 3 samples were negative for NiV RT-PCR and isolation precautions were discontinued. He was started on IVIG 2 gm/kg × 5 days for persistent dysautonomia [Figure 3].

From day 24, Echo showed persistent pulmonary artery hypertension [PAH], likely due to NiV pulmonary vasculitis [C-ANCA, P-ANCA and thromboembolic workup were negative] [Figure 2]. At discharge [day 52] he still had resting tachycardia, exertional dyspnea and cortical poly-mini-myoclonus.

After an incubation period of 5-14 days, NiV presents as an AES.^[2] Encephalitis or brainstem involvement indicates a poor prognosis.^[2,3] NiV causes an extensive small vessel vasculitis affecting most organs.^[4] Tiny [<5 mm] necrotic plaques are scattered in the white and grey matter accounting for the 'Starry sky' appearance on MRI.^[5,6] The term 'Patient Zero' refers to the index case in a communicable disease or the first incident in the onset of a catastrophic trend.

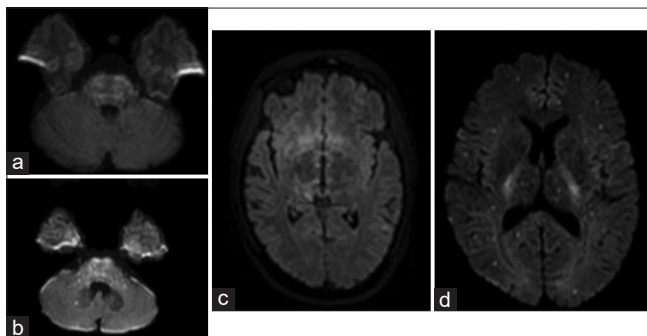


Figure 1: MRI Diffusion weighted image [DWI] sequences. Panel A shows tiny punctate lesions in the pons. Panel B shows pontine lesions extending into the middle cerebellar peduncels and a few lesions in the cerebellum. Panel C shows punctate lesions in the thalamo-hypothalamic area. Panel D shows the 'Starry sky' appearance of multiple cortical and subcortical hyperintensities

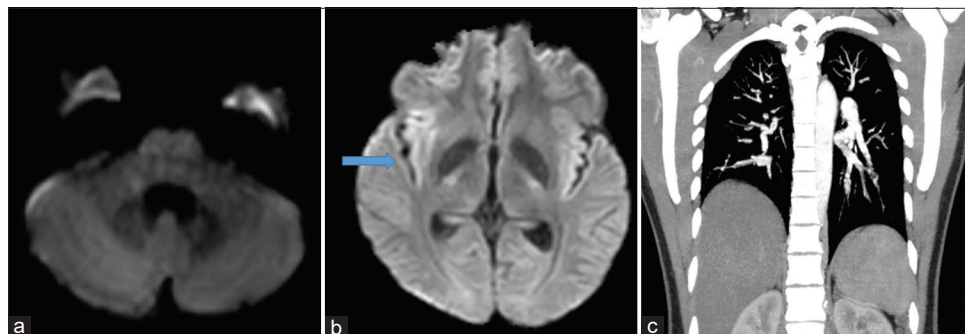


Figure 2: MRI DWI sequences. Picture on the left showing resolution of pontine DWI lesions. Picture on the right shows a right insular DWI hyperintensity. Panel on the extreme right, CTPA; shows normal pulmonary vasculature

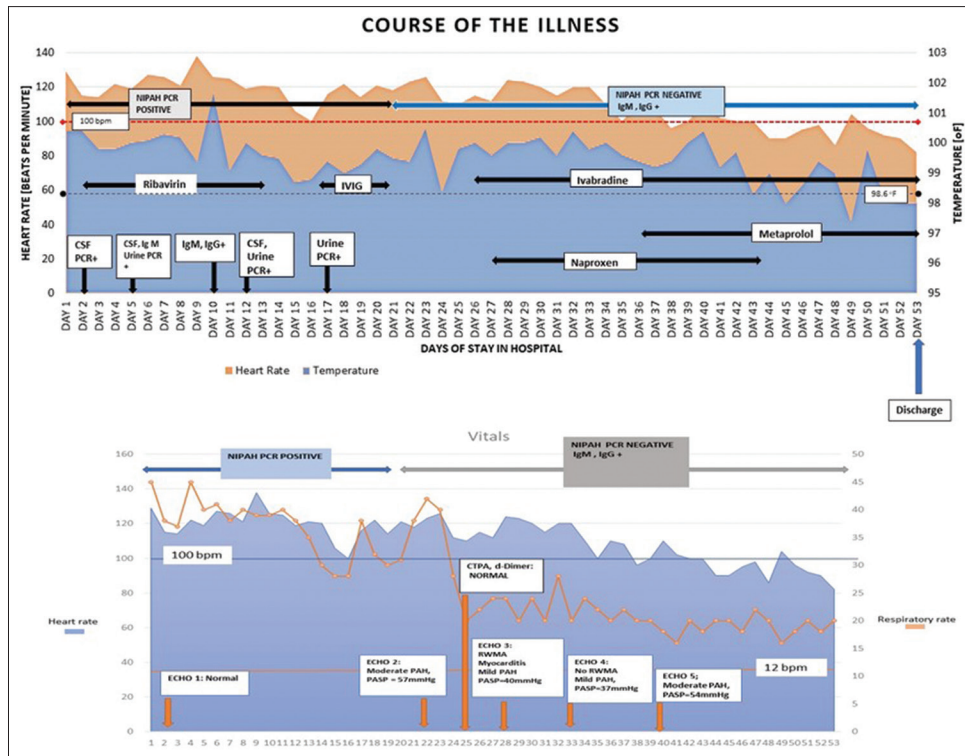


Figure 3: Shows the clinical features of the patient during hospitalization

In 2019, a NiV outbreak was stopped at ‘Patient Zero’ in Kochi; an unusual phenomena and moreover due to the protracted hospitalization, we could identify uncommon features of NiV infection such as dysautonomia, transient myocarditis and pulmonary arterial hypertension.

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Conflicts of interest

There are no conflicts of interest.

References

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